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Uterine Rupture Following Full-Term Vaginal Delivery: A Case Report

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Abstract

Uterine rupture in an unscarred uterus is an obstetric emergency that can lead to considerable morbidity and mortality for both mother and fetus. It is uncommon yet fatal. This case report presents a 39-year-old woman, gravida 4 para 3+0, with no prior history of uterine surgery or complications during her antenatal visits. At 38 weeks of gestation she delivered a healthy neonate via spontaneous vaginal delivery. Following the delivery, she suffered from massive postpartum haemorrhage and was unresponsive to initial management, including the use of uterotonics, balloon tamponade, and resuscitation. While transferring her to a tertiary care facility, the posterior uterine rupture was identified, and she underwent emergency surgical intervention. After resuscitation with fluids and blood products, the patient was monitored closely and underwent uterine repair. The case underscores the importance of considering uterine rupture as a possible cause of persistent postpartum bleeding, even without a prior cesarean section history. It highlights the need for prompt diagnosis, surgical intervention, and multidisciplinary care to optimize maternal health in such rare but high-risk case scenarios.

Keywords: Uterine rupture, postpartum haemorrhage, obstetric emergency, vaginal delivery

Introduction

Uterine rupture is a rare but potentially fatal obstetric complication often associated with an unscarred uterus (no previous cesarean or other uterine surgery). It is like the rupture of any internal organ that poses significant risks to both the mother and fetus, often resulting in severe morbidity and mortality. Rupture of the uterine wall, particularly in the absence of prior uterine surgery or trauma, is a rare occurrence, estimated to affect 1 in 5,700 to 1 in 50,000 pregnancies.[1] In a study of 75 cases involving complete uterine rupture (full disruption of the uterine muscle and serosa) in unscarred uteruses, the incidence was reported as 0.2 ruptures per 10,000 births. Among these cases, 18 ruptures occurred preterm, and 6 occurred before labor.[2] The overall incidence of uterine rupture in scarred and unscarred uteruses has increased in recent decades.[3] Ruptures of the unscarred uterus are more common in resource-limited regions, where timely access to the management of cesarean sections and assisted vaginal deliveries for managing obstructed labor is

often unavailable.[4] In unscarred uteruses, the risk of rupture can be affected by several factors. The most important of these would be prolonged or obstructed labor, since both can result in significant pressure on the walls to the point where there may be a rupture. Macrosomia, where the baby is too large, is likely to result in overstretching of the uterus during delivery, especially in cases of cephalopelvic disproportion. Likewise, higher parity is likely to cause stretching of the uterus, thereby increasing the probability of a rupture occurring. Furthermore, the injudicious use of oxytocin or prostaglandin agents can result in hyperstimulated uterine contractions, which increase the risk of rupture during labor. Other traumas, like falling or a car accident, may also cause damage to the uterus and result in a rupture during or after pregnancy.[5] The increased age of the mother can be another risk since older women tend to have poor tissue elasticity and other premorbid conditions that make the uterine wall weaker. Additionally, having placenta percreta or increta can cause abnormalities and reduce the strength of the myometrium, therefore increasing the risk of rupture. Having a short duration between pregnancies can also lower the strength of the uterine walls, particularly if the previous pregnancy caused substantial uterine distention. Delay in prenatal care is likely to cause problems like presentation or cephalopelvic disproportion, thereby increasing the probability of complications.[6]

Case Presentation

Patient Profile

A 39-year-old woman, gravida 4 para 3+0, was presented at 38 weeks of gestation. Her antenatal course was unremarkable, and she was admitted in active labor to a private secondary care hospital. She had no significant medical or surgical history, including no history of cesarean section or uterine instrumentation. Her previous deliveries were all spontaneous vaginal deliveries without complications.

Clinical Course

The patient delivered a healthy female neonate weighing 3 kg via normal vaginal delivery with mediolateral episiotomy given. However, a few minutes postpartum, she developed significant vaginal bleeding. Initial uterotonic agents, including oxytocin and misoprostol, were administered, and bimanual uterine massage was performed to address suspected uterine atony. A balloon tamponade was also in placed. Despite the measures, the bleeding persisted, and resuscitation was initiated with intravenous crystalloids and 3 packed red blood cell transfusions. Laboratory findings are summarized in Table 1. On examination, her Glasgow Coma Score was 15 out of 15, blood pressure was 97/64mmHg, heart rate was 121 beats/min, SPO2 was 98%, and she was afebrile. Her abdomen was soft, non-tender, and well-contracted. A Foley catheter was placed, and the uterus was noted to be well-contracted on palpation, ruling out uterine atony. Examination revealed no vaginal or cervical lacerations. With an ongoing hemorrhage, the patient was transferred to a tertiary care hospital for further management.

Table 1: Laboratory Findings

Parameters	Result	Reference Ranges
Haemoglobin (Hb)	5.6g/dl	11 - 14.5 g/dl
Haematocrit	17.2%	34.5 - 45.4%
W.B.C	36.7*10E9/L	4.6 – 10.8*10E9/L
Platelets	139*10E9/L	154 - 433*10E9/L
Prothrombin Time (PT)	11.3 sec	9.3 – 12.8 sec
Activated Partial Thromboplastin Time	36.1 sec	22.9 – 34.5 sec

Management at Tertiary Care Hospital

On arrival at the tertiary care hospital, she was actively bleeding vaginally. The tamponade was removed, and she was immediately taken to the operating theatre for exploration. Intraoperatively, around 600ml of haemoperitoneum was found, and a uterine wall rupture at the posterior wall of the uterus extending up to the cervix 3 to 4 cm was identified. The anterior uterine wall was intact, and both tubes and ovaries appeared normal. Left internal iliac ligation was performed. A total of 4 packed red blood cells, 2 units of platelets, and 2 units of fresh frozen plasma were transfused intraoperatively. The bleeding was controlled, and the patient was shifted to the Intensive Care Unit for post-op management. The laboratory findings of pre and post-blood transfusion are summarized in Table 2.

Table 2: Laboratory Findings

Parameters	Result	Reference Ranges
Pre-Transfusion Labs		
Haemoglobin (Hb)	6.2g/dl	11 - 14.5 g/dl
Haematocrit	17.9%	34.5 - 45.4%
W.B.C	16.2*10E9/L	4.6 – 10.8*10E9/L
Platelets	93*10E9/L	154 - 433*10E9/L
Post-Transfusion Labs		
Haemoglobin (Hb)	10.9g/dl	11 - 14.5 g/dl
Haematocrit	32.8%	34.5 - 45.4%
W.B.C	17.1*10E9/L	4.6 – 10.8*10E9/L
Platelets	120*10E9/L	154 - 433*10E9/L
Fibrinogen Level	211mg/dl	156 – 400 mg/dl
Prothrombin Time (PT)	10.9 sec	9.3 – 12.8 sec

Postoperative Management

The patient was monitored in the Intensive Care Unit for 48 hours postoperatively. Her recovery was uneventful, with stable haemoglobin levels and no further bleeding. She was discharged on the third postoperative day with detailed counselling on future pregnancy risks, including the possibility of elective cesarean delivery to avoid recurrence.

Differential Diagnosis

1. Uterine Atony- Failure of the uterus to contract adequately post-delivery, leading to massive postpartum haemorrhage.
2. Placental Abruption- Premature detachment of the placenta from the uterine wall, causing pain and bleeding.
3. Cervical or Vaginal Lacerations- Tears in the birth canal that can result in significant bleeding, mimicking uterine rupture.
4. Inverted Uterus- A rare condition where the uterus turns inside out, often leading to severe pain and haemorrhage.
5. Retained Products of Conception (RPOC) - Retention of placental or fetal tissue, causing uterine bleeding and preventing contraction.

Management of Uterine Rupture and its treatment plan

1. **Resuscitation.** IV crystalloids, such as normal saline, were administered to restore circulatory volume. Administer blood components like packed red blood cells, fresh frozen plasma, and platelets according to the level of haemorrhage and test results of the patient.
2. **Oxygenation and Monitoring.** Supplemental oxygen is used to maintain adequate oxygen saturation and continuously monitor vital signs, urine output, and hemodynamic stability.
3. **Surgical Intervention.** Surgery was done immediately, i.e., exploratory laparotomy, was performed to determine the cause of the bleeding.
4. **Postoperative Rehabilitation.** Gradual diet progression to provide healing. Physiotherapy to reestablish mobility and inhibit complications.
5. **Family Education and Counseling.** Her family acknowledged counseling about her medical condition, regular monitoring, and follow-up care during the patient's hospital stay.
6. **Outcome.** The patient showed prominent clinical progress and was positively discharged on the third postoperative day.
7. **Pharmacological Therapy.** Numerous medications were directed as part of the patient's treatment plan. Table 3 summarizes the key drugs used in the patient's management plan.

Table 3: Pharmacological management of the patient

Drugs	Classification & Mechanism of Action
Oxytocin	Classification: Uterine Stimulants Mechanism of Action: Stimulate uterine smooth muscle contractions to control bleeding.
Tranexamic Acid (TXA)	Classification: Antifibrinolytic Mechanism of Action: Inhibits fibrinolysis, stabilizing blood clots to reduce bleeding.
Ceftriaxone	Classification: Broad-spectrum antibiotic Mechanism of Action: Prevents or treats bacterial infections.
Colistimethate Sodium	Classification: Antibiotic Mechanism of Action: Interrupts the bacterial cell membrane by binding to lipopolysaccharides and phospholipids, primary to cell death.
Omeprazole	Classification: Proton pump inhibitor Mechanism of Action: Inhibits the enzymes, reducing gastric acid secretion.
Paracetamol	Classification: Analgesic and Antipyretic Mechanism of Action: Inhibits prostaglandin synthesis and modulates the hypothalamic heat-regulating center to reduce fever. Provides pain relief.

Discussion

Uterine rupture is the complete division of all three layers of the uterus, i.e., endometrium, myometrium, and perimetrium.[9] In an unscarred uterus, it is an uncommon but potentially fatal obstetric emergency that requires heightened vigilance, especially when postpartum haemorrhage fails to respond to conventional treatments.[7] Uterine ruptures are commonly associated with risk factors such as a history of prior cesarean deliveries or uterine surgeries. Other factors include

prolonged labor, fetal macrosomia, high parity, advanced maternal age, and the over usage of uterotonic drugs like oxytocin, also predispose to uterine rupture in an unscarred uterus.[8] Low socioeconomic status, poverty, uncontrolled birth rates, lack of awareness, and less gap in deliveries are other risk factors of uterine ruptures. While the incidence of unscarred uterine ruptures is low, its rate is rising.[4] Uterine ruptures occur at varied percentages worldwide.[10] The rate has drastically decreased in developed countries. Although it remains one of the main health challenges in developing countries like Pakistan. Its frequency in Pakistan is 1.05%, whereas in the northern areas of Pakistan, the rate is 12.5%.[10]

Considering uterine rupture in this case was unexpected because the patient had a history of all normal spontaneous vaginal births and exhibited no symptoms of previous uterine trauma. A thorough evaluation of the differential diagnosis was necessary because the clinical picture showed characteristics of uterine rupture, accompanied by ongoing vaginal bleeding and without symptoms of uterine atony. In such cases of unresolved postpartum bleeding, the patient's unstable hemodynamics and continuous haemorrhage validated how critical it is to identify uterine rupture. First-line therapy for uterine atony, such as balloon tamponade, bimanual massage, and uterotonic drugs, were used to treat the patient. The lack of improvement, however, suggested a different underlying problem, such as uterine rupture. The decision to move forward with surgical exploration was essential because it made it possible to locate and fix the uterine rupture. Obstetricians, anesthetists, and intensivists worked together in a multidisciplinary manner to control the bleeding and stabilize the patient's condition. Additionally, prompt surgical intervention is crucial in the therapy of uterine rupture. The patient needed a transfusion of blood products such as packed red blood cells, fresh frozen plasma, and platelets to address the hypovolemia and coagulopathy after the rupture was successfully healed.

In cases of refractory postpartum hemorrhage, especially in women who have no previous uterine surgery, this case highlights the necessity for increased observance. It also emphasizes the significance of antenatal care, which includes determining risk factors that could make issues like uterine ruptures more likely.

Conclusion

Uterine rupture in an unscarred uterus, even though uncommon, needs to be taken into account when making a differential diagnosis for postpartum haemorrhage. Early detection and surgical intervention are required for preventing severe maternal morbidity and mortality and for achieving healthy outcomes.

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